BULLETIN OF THE

PET PRACTITIONERS ASSOCIATION OF MUMBAI

(FOR CIRCULATION AMONGST PPAM MEMBERS)

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Contents

Dr. S. V. Vishwasrao
Ph.D. (Surgery)
Editor, PPAM Bulletin
vishwasraodr@hotmail.com
Mobile : 9322242184

Editorial

A New Generation of Students: PPAM Members Come Forward as Speakers

Hon Dr. Abdul Kalam sir famous quote "Dream is not something which you see in your sleep but is one which does not let you sleep". All budding Veterinarians in final year of B.V.Sc & A.H. have a dream of what they want to pursue after graduation. The outreach program planned by PPAM in collaboration with MAFSU is step to guide the young dreamers in that direction.

Young veterinarians constitute majority of our veterinary professionals. Our young budding veterinarians also come from varied and complex background, and grappling with a range of personal, professional and carrier challenges. All these issues have a component for professional development, with linkages to education, gender equality, constructive behaviours, professional bonding, personality development, confidence to practice veterinary medicine and achievement of sustainable development goals in their personal and professional life.

In veterinary education one young veterinarian teaching other called as peer learning is an education method that helps students solidify their knowledge. One young veterinarian tutoring another in a supervised environment can result in better learning and retention. Because to teach another, the first veterinarian must first fully understand a concept themselves.

Veterinary colleges many a times operate on tight budget and unable to invite expert faculty from our country or foreign faculty or experts in particular field. PPAM needs to step in and fill this void to some extent by organizing out reach programs in veterinary colleges.

While veterinary colleges in India have a reasonable fee structure, they suffer from underfunding and paucity of teaching staff. Private professional organizations try to fill this gap, but often at a price that is too steep for young budding veterinarians.

Amongst PPAM members there are speakers and experienced practitioners who can contribute to this upgrading program. PPAM would love to invite these PPAM members to be a part of this team to address students on topics relevant and interesting to these students.

You need to identify your core area of specialization, its relevance to final year students of today and make a power point presentation for 25-30 minutes, be able to answer any questions from students and make an impact on the students. This is going to be challenging but I am sure many of our both young and experienced veterinary members will come forward for this noble task which would be satisfying and fulfilling. PPAM would love to invite speakers from different fields, depending on feedback we get from students, and speakers' abilities to prepare ppt presentation addressing local veterinary problems and respecting local cultures.

The backbone of these outreach programs will be final year students and speakers. All the speakers invited will preferably come from different streams of veterinary profession, so, they come and share their experiences with students, how their veterinary carrier shaped and how they overcame difficulties. The final year students will get to know a lot more about different facets of our profession not usually taught in our syllabus.

Interested PPAM member of sharing their knowledge as speakers for the outreach program may contact the editor PPAM bulletin on mobile number, WhatsApp number or email mentioned in this bulletin.

11 FASAVA CONGRESS

October 27 - 29, 2023 • Venue - Mumbai, India

Dr. Jairam Ramani, PPAM FASAVA Representative, Executive Board Member & CPD Director FASAVA

PPAM hosted the 11th FASAVA Congress at The Westin, Powai.

The event was attended by over 1300 veterinarians from across Asia. We had veterinarians from Australia, Japan, Malaysia, Taiwan, Sri Lanka, Nepal, South Korea and India. This was biggest gathering of veterinarians to be held in India. The Congress was held simultaneously across 7 halls covering a wide range of topics from Emergency and Critical Care, CBVD, Cardiology, Dermatology, Exotic Animal medicine, Gynaecology, Imaging and Frline medicine. A large animal stream was held for the first time at a FASAVA Congress. A successful Poster presentation competition was also held during the Congress.

This 3 day event marked an important milestone in the history of PPAM.

The event was inaugurated by VCI President Dr. Umesh Chandra Sharma. The event started with the rendition of the Ganesh Vandana and the traditional lamp lighting ceremony. This was followed by singing of our national anthem. The Flag handover ceremony was conducted. The FASAVA President Dr. Matthew Retchford handed the FASAVA flag to PPAM President Dr. Dhananjay Bapat.

Dr. Umesh Chandra Sharma gave a nice speech stressing the importance of quality veterinary education and the role of veterinarians to our Society.

The first Dr. Roger Clarke plenary session lecture was delivered Dr. Khorshed Mama from Colorado State University (USA). The hall was packed to capacity with over 900 delegates attending the plenary session.

The 132 lecture schedule was amazing with 7 lecture streams running simultaneously.

Each session had a moderator and a timer was displayed to keep track of time. The LED screens were used in all the halls. The AV arrangements were excellent. All the notes were handed over to the participants on a pen drive.

There were 41 speakers who delivered world class lectures. The congress also saw a good interaction between the speakers and the delegates. The quintessential Indian veterinarian asking all the questions and finding all the correct answers. Emerging fields were thrown open to the practicing veterinarians.

The 11th FASAVA Congress saw generous support from the sponsors. Special mention of Drools, Royal Canin, Pedigree, Zoetis and MSD. The industry exhibits were very tastefully decorated and saw 40 plus stalls.

We had cake cuttings, coffee servings and lucky draws being held during the 3 days

Dr. Khorshed Mama delivered the Dr. Roger Clarke Plenary lecture on the 27th October setting the tone for the FASAVA Congress. Dr. Angela Lennox led the way in exotic mammalian medicine. This is the field that has seen the fastest growth in the past 6 years. Dr. Vellayan Subramanian taught the basics of turtle care. Dr. Eliza Mazaferro was at her best teaching the rules of Emergency and Critical Care. Dr. Meg Sleeper and Dr. Angela Lennox covered Cardiology and Common vector borne diseases. Dr. Takou Ishida and Dr. Han Hock Siew covered Pathology and Dermatology. Dr. Joerg Steiner covered Intestinal Dysbiosis and various ailments of the Gastrointestinal tract. Dr. Laura Selmic covered oncology and onco surgery.

A large animal stream was held was the first time at a FASAVA Congress. It was much appreciated. Dr. Rajendra Dhumal, Dr. Phiroze Khambatta, Dr. S. Upadhye, Dr. Dayaram Suryawanshi, Dr. Gatne M. L., Dr. Markandeya, Dr. R. K. Jadhav did our nation proud.

All the Indian speakers were well appreciated and their understanding and grasp of their subject helped the delegates. The sessions were well moderated and time was maintained in all the sessions.

Cultural programs featuring Indian dance forms. There were 2 gala dinners organised. The poster session was held in the Lotus hall. The judging of the poster was by an international jury. Dr. Deepti Deshpande, Dr. Prathamesh Deshmukh, Dr. Noopur Desai, Dr. Pushkar Banka and Dr. Aboli Lakhpati were the winners.

All in all it was very successful Congress. The 12th FASAVA Congress will be held in Kualalumpur, Malaysia from 19-21 July 2024.

11 FASAVA CONGRESS


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Stop Rabies Campaign

A partnership between PPAM, MCGM (Municipal Corporation of Greater Mumbai) and Boehringer Ingelheim India for the Stop Rabies Project.










STOP RABIES PROGRAM

A Partnership between PPAM (Pet Practitioner Association of Mumbai), MCGM (Municipal Corporation of Greater Mumbai) and Boehringer Ingelheim India for the Stop Rabies Project that aims to vaccinate uncared dogs and cats and drive awareness around rabies. Both PPAM and Boehringer Ingelheim India, firmly believe that long-term local action is key to making a lasting impact on the Stop Rabies Program. PPAM & BI thank Welfare of Stray dogs (WSD) wholeheartedly for outreach to many school children for awareness about Stop Rabies program.

THE QUILTRO

Dr. Rashmi Gokhale, Centre for Animal Population Studies

A *quiltro* means a street, mixed-breed dog in Chile. A fossil of a jaw bone of canine, dating back 12,000 years ago is found in Central America closer to Chile, South America. The piece of evidence suggested that dogs were living with their master (human) in Costa Rica in the Pleistocene era[1]. Christianity is a major religion in Chile[4]. Modern Chilean urbanization is a mix of Spanish western naturalism and ancient American animism[4].

Companion animal protection laws in Chile are ranked 'D' by the WAP Animal Protection Index[4]. Further, there is no specific legislation relating to street animals in Chile. Law 20380, the main animal protection legislation in Chile, establishes the duty of care by companion animal owners and holds them responsible for their animal's action in society[3]. Municipal Corporation is responsible to control the companion animal population to protect the environment and public health under Law 21020[3].

The curious case of community dogs

A survey of dog owners in 12 rural areas in the Paine municipal limits was performed to study domestic dog ownership[2]. 85% of the households owned at least one dog, contributing to the human: dog ratio of 1.5:1. Majority of the surveyed dogs were mixed-breed, male and 1 to 7 years old. Out of those 63.1% males, only 3.1% were castrated[2].

Free-roaming dogs in Plaza Peru were observed using crosswalks alone or with fellow dogs or humans while crossing the busy streets[4]. A free-roaming dog could be a family dog or restricted dog or street dog or feral dog[11]. Miternique and Gaunet have categorized free-ranging dogs in 9 sub-classes to study their behavior based on spatial differences. A free-roaming-pet or community dog is more familiar, less stressful and almost not fearful to humans, increasing the chances of proximity to unfamiliar human beings[4].

Most of the dog owners in Paine disliked free-roaming dogs and preferred to confine their own dogs. However, the owners failed to comply with their say to confine their dogs[3].

Welfare assessment of a community *Quiltro*

We will use the Five Welfare Needs framework to assess welfare of an intact, male, Chilean community dog below as an example[12]. Further, we will attempt to find solutions to the welfare concerns using Adequate Guardianship framework. The framework addressed welfare needs of dogs, namely, food, water, basic veterinary care, safe & appropriate shelter, avoidance of injury or cruelty, exercise, and companionship.[7]

Addressing the identified welfare needs

1) Nutrition & Physical health

a) Diet[7]

Dog is facultative carnivore and conditional omnivore. A community dog is less likely to receive commercial pet food for all his meals, causing nutritional deficiency. Full commercial diet could be nutritionally enriching but mentally non-stimulative. House scraps, bone pieces, small wild prey etc. found during roaming act as enrichment. A diet for community dogs shall be designed to be nutritionally sound and economical.

b) Body Condition Score (BCS)[5]

BCS, a good representation of nutritional status, can be measured from 1 to 5, 1 being easily visible ribs to 5 being ribs non-palpable and hidden under a thick layer of skin.

2) Environment (resources)

a) Basic necessities- Food, water and Shelter

Owned dog is a dog for which a person claims responsibility[11]. Therefore, WOAH guides the guardian to provide adequate fresh water, nutritious food, clean and hygienic environment to the dog. Routine inspection and monitoring for health status are equally important. WOAH guides the owners to follow *responsible dog ownership*[11]. Community dogs are semi-owned dogs.

A suitable shelter provides protection from hazards, a safe & comfortable place to rest in a dry, heat-proof

area, a place to hide and a toilet area.[12] It is essential to identify whether these needs are met in a free-roaming-owned or community dog.

b) Basic veterinary care

A free-roaming dog should receive timely preventive medication and a good death. Vaccination, sterilization, and euthanasia are the three key aspects of basic veterinary care[10]. Male, owned/ownerless, free-ranging dogs are difficult to restraint, a major limitation in delivering basic veterinary care[10].

Canine parvovirus, adenovirus and distemper virus are common enzootic dog pathogens in India against which vaccination is routinely carried out in the veterinary practice. These infections are zoonotic, affecting local wildlife too[9].

Catch-Neuter-Vaccinate and Return (CNVR) is a method to reduce the free-ranging dog population. A good euthanasia policy, humane and scientific shelters, and behavioral training of dogs at the shelter can not only reduce dog population but also improve welfare of dogs[12].

3) Behavior & Mental health

A) Activity [6]

To understand the intrinsic behavior of dogs without direct human intervention, Free-ranging domestic dogs (FRDD) and family dogs were fitted with tracking devices in a study to measure their activity. FRDD were most active during the morning and late afternoon. Whereas activity patterns in family dogs vary with owner's routine and activity.

B) Social interaction[9]

A territorial range of male domestic dog is more than a female dog due to sex and reproductive influence. An intact male may not prefer to socialize with spayed female. On the contrary, a castrated male is well accepted by intact female in a territory. Even though Owned dogs are allowed to roam freely like any other family member. But they limit their interactions within the home community.

Take home message- Supervise and Sterilize

Free-roaming, semi-owned-dogs welfare status depends upon humans in that locality. Dogs are extremely adaptable, both socially and geographically. Street-owned dogs can prey on small-sized livestock and wildlife, which create conflict with humans and the environment. Zoonotic disease spillover may not be eliminated by vaccination of street dogs. Well supervised activities of pet dogs and controlling free-ranging dog population through sterilization are inevitable solutions. A community-based intervention in terms of low-cost spay-neuter clinics, participation of local communities, policy makers and veterinarians can ease the management of community dogs for optimum welfare.

If the caregiver is facing challenges to confine an intact, male community dog due to his straying behavior, then castration can be considered as one solution. Castration, an integral duty of care of the caregiver, makes the male dog sexually inactive, reducing the territorial behavior and consequent dog fights. But it cannot be considered the only solution to control the street dog population.[12]

References

1. 12 Oct 2021. Humans and Dogs 'lived together 12,000 years ago'. *Free Malaysia Today*. Available at: https://www.freemalaysiatoday.com/category/top-lifestyle/2021/10/12/humans-and-dogs-lived-together-12000-years ago/?__cf_chl_jschl_tk__=pmd_LgnT1Dk718zy0gjK9_gWCJvHE.IygpjYouRMr8e32x8-1634713938-0-gqNtZGzNAqWjcnBszQfl [Accessed on 20 Oct 2021]
2. Astorga, F., Poo-Munoz, D. A., *et.al*. 2020. Why let the dogs out? Exploring variables associated with dog confinement and general characteristics of the free-ranging owned-dog population in a per-urban area. *Journ of Appl. Anim. Wel. Scie.* DOI: 10.1080/10888705.2020.1820334.
3. 2020. Chile- Animal Protection Index. *World Animal Protection*. Available at: https://api.worldanimal protection.org/country/chile
4. Miternique, H. C. & Gaunet, F. 2020. Coexistence of diversified dog socialities and territorialities in the city of ConcepciÓn, Chile. *Animals.* 10:298. DOI: 10.3390/ani10020298
5. Dinkelmeyer, A. 2013. How IFAW helps dogs and cats. *IFAW.* Available at: https://s3.amazonaws.com/ifaw-pantheon/sites/default/files/ legacy/IFAW-Animal-Approach-Update.pdf [Accessed on 23 Oct 2021]
6. Griss, S, Riemer, S, *et.al*. 2021. If they could choose: how would dogs spend their days? *Appl. Anim. Beh. Scie.* 243: 105449. Available at: https://doi.org/10.1016/j.applanim.2021.105449
7. Stafford, K. 2007. Chap 4. Canine nutrition and welfare in the *The Welfare of Dogs*. Springer. Vol 4: 83. ©ISBN 978-1-4020-6144-8.
8. Belsare, A.V. 2013. Disease ecology of free-ranging dogs in Central India: Implications for Wildlife Conservation. *ProQuest Dissertations & Theses Global*. ©ISBN: 9781369294569.
9. Sparkes, J., Kortner, G., *et.al*. 2014. Effects of sex and reproductive state on interactions between free-roaming domestic dogs. PLoS ONE 9(12): e116053. https://doi.org/10.1371/journal.pone.0116053.
10. OIE. Chap. 7.7. Stray dog population control. *Terrestrial animal health code*. Available at: https://www.oie.int/fileadmin/Home/eng/Health_standards/tahc/2018/en_chapitre_aw_stray_dog.htm [Accessed on 21 Oct 2021]
11. Dept. of Environment Food and Rural Affairs. 2018. Code of Practice for the welfare of dogs. Govt. of UK. © Crown copyright. Ref: ISBN 978-1-5286-0171-9

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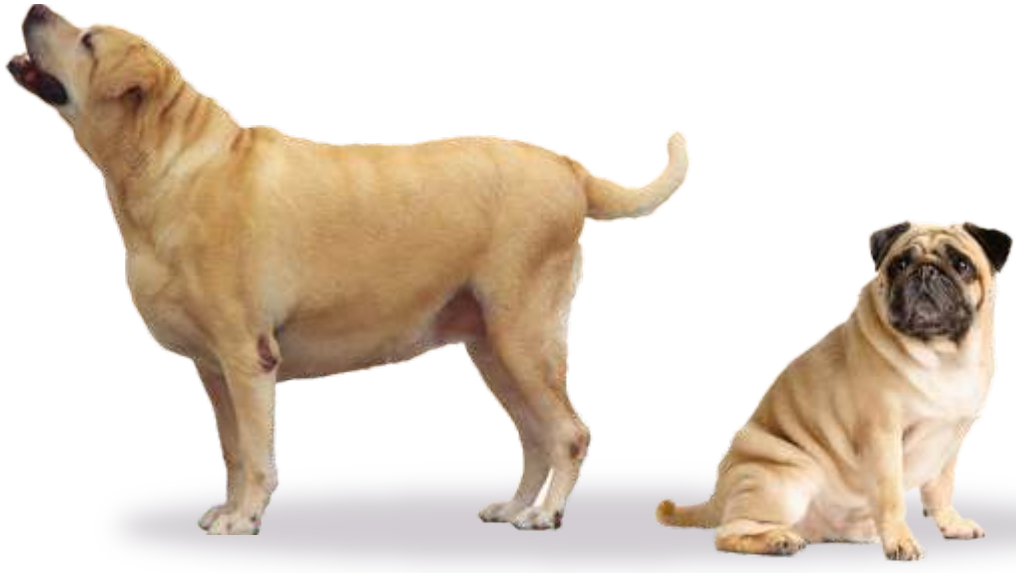


SCAN TO KNOW MORE

DROOLS ARTICLE

Cardiometabolic Conundrum: Unraveling the Complex Nexus Between Obesity and Heart Diseases; A review

Dr. Jadhav Aditya Sudhir
Veterinary Product Executive (Specialist)

Obesity arises when a pet's energy intake surpasses daily expenditure, often due to reduced exercise and overfeeding. A dog weighing 20% or more above normal is clinically obese. This health issue, prevalent in both humans and pets, is alarming globally, with about 56% of dogs and 60% of cats identified as obese (APOP, 2017).

The most common cause of heart failure in dogs is valvular disease, especially in small breeds, and it accounts for 75% to 80% of canine cardiac disease[1]. Myocardial disease, especially dilated cardiomyopathy, is also common in dogs[2]. Cardiomyopathies, especially hypertrophic cardiomyopathy (HCM), are the most common causes of heart disease in cats, accounting for more than 60% of feline heart disease[3]. Obesity does not increase the risk for atherogenic coronary heart disease in dogs and cats as it does in humans. Atherosclerosis is rare in dogs and extremely rare in cats, likely in part due to their relatively high high-density lipoprotein (HDL) to low LDL ratio[4]. Obese cats do have an increase in VLDL's similar to humans but do not develop subsequent hypertension and atherosclerosis, so other antiatherogenic mechanisms may be present[5,6]. Obese dogs show hypertriglyceridemia and hypercholesterolemia, but most of the cholesterol is HDL cholesterol, which is protective against atherosclerosis. The ratio of HDL to LDL cholesterol does not change with weight gain in dogs as it does in humans[7]. Obesity at the time of diagnosis has not been reported as a risk factor in epidemiologic studies of heart disease in cats and dogs. In 1 study, the median body condition score (BCS) of the dogs with chronic valvular disease was 6 (range of 4–9) whereas the control dogs had a median BCS of 5 (range of 5–7). This difference was not significant (P= 0.37)[8]. In another study comparing dogs with heart disease and healthy dogs, there was again no difference in BCS. This study did find an increase in the amount of visceral abdominal obesity in the dogs with heart failure compared with healthy dogs[9]. In more than 50% of dogs with dilated cardiomyopathy, cardiac cachexia (a loss of lean body mass) was reported[10]. Body weight was not a risk factor in a study of feline HCM[11], and BCS was also not different for cats with HCM compared with control cats, although the cats with HCM were skeletally larger[12]. Because obesity is common in adult dogs and cats, an overweight or obese body condition may have been present previously in pets with heart failure, because weight loss is a common presenting sign; however, this still does not qualify obesity as being a risk factor.

The Effect of Obesity on Heart Function

Obesity is known to have deleterious effects on heart function. Obese dogs have an increase in heart rate and a small to moderate increase in blood pressure compared with lean dogs[13–16]. The increased heart rate may be due to reduced parasympathetic control of heart rate[13], and the increased blood pressure due to an altered sympathetic control of peripheral vascular resistance[14]. Compared with lean dogs, obese dogs also have increased left ventricular free wall thickness at end diastole and end systole.

Renin-Angiotensin-Aldosterone System

As well as its role in blood pressure control, the renin-angiotensin system (RAS) plays a role in adipocyte differentiation and metabolism. All components of the RAS are found within fat, and white adipose tissue is a major source of angiotensinogen, second only to the liver[17]. Obesity in humans, rodents, and dogs is associated with activation of the RAS as evidenced by increased levels of circulating angiotensinogen, plasma renin activity, angiotensin-converting enzyme activity, angiotensin II, and aldosterone[18–20]. Increased abdominal visceral fat is associated with a more pronounced activation of the RAS compared with peripheral subcutaneous fat[17]. Overweight dogs have a higher systolic blood pressure than normal weight dogs[7,21,22], although the difference is not always significant[21]. Obesity has not been reported as a risk factor for hypertension in cats. With increased fat mass, adipose-derived angiotensinogen increases plasma angiotensinogen concentrations, resulting in more angiotensin II. Increases in angiotensin II promote cardiovascular dysfunction by direct vasoconstrictor activity and by increasing renal sodium retention via increased aldosterone release, which can increase blood pressure[18–20]. Aldosterone

and renin have been reported as elevated in dogs with obesity-induced hypertension that had been fed on a high-fat diet for 5 weeks[23]. Using eplerenone to antagonize the aldosterone decreased hypertension in these dogs. It is also possible that the high-fat diet contributed to hypertension because high-fat diets can cause sodium retention in some species[24]. Weight loss in humans decreases circulating components of the RAS[25], and weight loss has been shown to decrease blood pressure in obese dogs[26]. As well as its effects on the vasculature, aldosterone is a mediator of inflammation, fibrosis, and oxidative stress and is involved in pathologic cardiac remodeling. Serum aldosterone and plasma renin are often elevated in dogs with congestive heart failure due to mitral regurgitation or dilated cardiomyopathy and in cats with restrictive or HCM. The production of RAS components by adipocytes is exacerbated during obesity[27]. In obese rats, the imbalance of the RAS system is thought to be due to oxidative stress[28]. Like rats, obese dogs have increases in oxidative stress[29], although the situation is less clear in cats[30].

Adiponectin

The effects of the adipokine (adiponectin) include improved insulin sensitivity and anti-inflammatory properties. It is generally decreased with obesity and increased in lean individuals. There has been some controversy about the decreasing of adiponectin in obese dogs. Some studies have shown a decrease in adiponectin in obese dogs[31-33]. It was speculated that obesity might decrease adiponectin in intact but not in neutered dogs[34]. Adiponectin has been shown to be decreased in obese cats[35]. Decreases in adiponectin in humans is more severe with increased visceral abdominal fat than with increased subcutaneous fat[17], and the adiponectin gene expression in cats is also greater in visceral compared with subcutaneous fat[35]. The decrease in adiponectin with increased fat, especially visceral fat, may be due to increased proinflammatory cytokines and increased reactive oxygen species, although in 1 study on canine adipocytes there was little effect on adiponectin production with the application of the inflammatory mediators TNF-a and lipopolysaccharide[36]. Adiponectin affects cardiovascular function and may have a palliative effect on pathologic cardiac remodeling after myocardial infarction[37]. The cardiovascular effects may result from the vasodilator functions of adiponectin[38]. Adiponectin increases expression of endothelial nitric oxide synthase and prostacyclin synthase, both of which promote vasorelaxation[39] and decrease the cardiac afterload[40]. Adiponectin also has anti-inflammatory and antiatherogenic properties, which may occur via its ability to suppress TNF-a production by macrophages, suppress myelomonocytic progenitor cell growth, and induce apoptosis[41].

Obesity Effect on Heart Size and Rate

Eighteen neutered female dogs that gained weight on a high-fat diet for 12 weeks developed concentric cardiac hypertrophy, although in the trial period there was no evidence of systolic or diastolic dysfunction, possibly due to the short duration of the obesity[42]. The cardiac hypertrophy in these dogs was reversed within 13 weeks of weight loss. The dogs, which were exercised, had less decrease in cardiac hypertrophy compared with those without exercise; however, exercise is known to stimulate physiologic ventricular hypertrophy. A study of 8 neutered beagles also showed an increased left ventricular wall thickness after weight gain, which was especially associated with visceral fat[21].

Weight Loss Effects on the Heart

Heart rate also increases with weight gain and normalizes after weight loss[15,42,43]. The increase in left ventricular wall thickness due to obesity also decreases with weight loss in dogs[21]. Owners often report a subjective improvement in activity after overweight dogs lose weight. In 15 previously overweight dogs, there was an increase in voluntary physical activity of 1.5 hours per day after weight loss[15]. Another study of overweight beagles showed a significant improvement in distance walked during the 6 minute walk test[44]. These dogs also had a subjective improvement in demeanor after weight loss.

The Obesity Paradox and Survival Times

Because of the increased risk of heart disease in humans, it seems that obesity would be associated with a worse survival rate for canine and feline heart failure patients; however, studies have shown that obesity may be associated with better outcomes after a diagnosis of heart failure, a phenomenon, termed the obesity paradox or reverse epidemiology. 2 Several large human studies have looked at the impact of BMI on mortality in heart patients; most have shown a significant improvement in survival with increased BMI, although in 1 study the benefit

was noted at 1 and 2 years but not at a 5-year follow-up45. Some studies have shown that in more marked obesity (BMI >35 kg/m2) there was no improvement in survival46. Nearly all studies show an improvement in overweight compared with cachexic patients. In dogs and cats, obesity does not increase the risk of coronary heart disease, but, as discussed previously, it can have an adverse effect on cardiopulmonary function and blood pressure[47], so weight loss may be recommended for obese dogs with increased risk of heart failure, such as high-risk breeds. One study in dogs with heart failure that gained, lost, or maintained body weight showed a difference in survival among dogs, with the dogs that gained weight surviving the longest[47]. Oddly, in this study, dogs that lost weight also lived longer than those who maintained their weight. The emaciated dogs with a BCS of 1 to 2 of 9 had the shortest survival time, with the obese dogs (BCS 8–9/9) living the longest; however, there were few dogs in these bottom and top categories and the comparison between BCS and survival was not statistically significant. The overweight dogs (BCS 6–7/9) did outlive the ideal (5/9) BCS dogs. A study of 101 cats with heart failure due to cardiomyopathy showed that cats with the highest and lowest body weights had reduced survival times compared with those with intermediate body weight. There was no statistically significant association between BCS and survival even though the pattern was similar to that for body weight[48]. In some human studies, moderate overweight body condition seemed to confer a survival benefit for heart disease, but patients with morbid obesity (BMI >35 mg/ kg2) had worse outcomes than those of normal weight or mild obesity[49]. A U-shaped curve by BMI may exist, similar to that in the feline study, where mortality is greatest in cachectic patients, lower in normal weight or mildly obese, and higher again in severely obese[49]. Obese human heart patients may be diagnosed earlier due to dyspnea, edema, or other respiratory signs or poor exercise tolerance, which may not be due to severe heart failure. This could mean that these obese patients have healthier hearts than nonobese patients at the time of diagnosis, leading to an apparent longer survival[46]. Lower BMI and a lower BCS in dogs and cats may reflect the presence of comorbid diseases, which could shorten survival time[49]. These comorbidities contribute to a state of malnutrition and shorter survival. Those patients who maintain or gain weight after diagnosis of their disease may be those who have a better response to treatment or fewer comorbid diseases. The neurohormonal alterations in obesity, for example, plasma adrenaline and renin concentrations, may favor a longer survival in someindividuals[45].

Obesity can lead to significant changes in cardiac structure and function, even in the absence of systemic hypertension and underlying heart disease. The increased total blood volume associated with obesity creates a state of elevated cardiac output, potentially resulting in ventricular dilatation and ultimately leading to eccentric hypertrophy, particularly in the left ventricle (LV) and possibly the right ventricle (RV) as well. Eccentric left ventricular hypertrophy, in turn, contributes to diastolic dysfunction, while systolic dysfunction may occur if wall thickening fails to keep pace with ventricular dilatation, a condition known as obesity cardiomyopathy.

The coexistence of systemic hypertension in obese individuals further promotes the development of left ventricular dilatation and hypertrophy.

Table 1. Comparison of Cardiac Structural and Hemodynamic Alterations in Patients with Morbid Obesity Alone, Systemic Hypertension Alone, and Combined Obesity and Hypertension

Variable	Obesity Alone	Hypertension Alone	Obesity and Hypertension
Heart rate	N	N	N
Blood pressure	N	↑	↑
Stroke volume	↑	N	↑
Cardiac output	↑	N	↑
Systemic vascular resistance	↓	↑	N or ↑
LV volume	↑	N	↑
LV wall stress	N or ↑	N or ↑	↑
LV hypertrophy	Eccentric	Concentric	Hybrid
LV diastolic dysfunction	Usually present	Usually present	Usually present
LV systolic dysfunction	Occasionally present	Usually absent	Occasionally present
LV failure	Occasionally present	Occasionally present	Commonly present
RV hypertrophy	Occasionally present	Usually absent	Occasionally present
RV enlargement	Occasionally present	Usually absent	Occasionally present
RV failure	Occasionally present	Usually absent	Occasionally present

LV = left ventricular; N = normal; RV = right ventricular; ↑ = increased; ↓ = decreased.

Consequently, congestive heart failure may manifest in these individuals, attributed to either left ventricular diastolic dysfunction or a combination of diastolic and systolic dysfunction. Systemic hypertension is present in 60% of obese individuals, and is severe in 10% and mild to moderate in the remaining 50%. Systemic hypertension predisposes to concentric LV hypertrophy, whereas obesity predisposes to eccentric LV hypertrophy. The coexistence of systemic hypertension and obesity has a greater effect on LV structure and functioning than

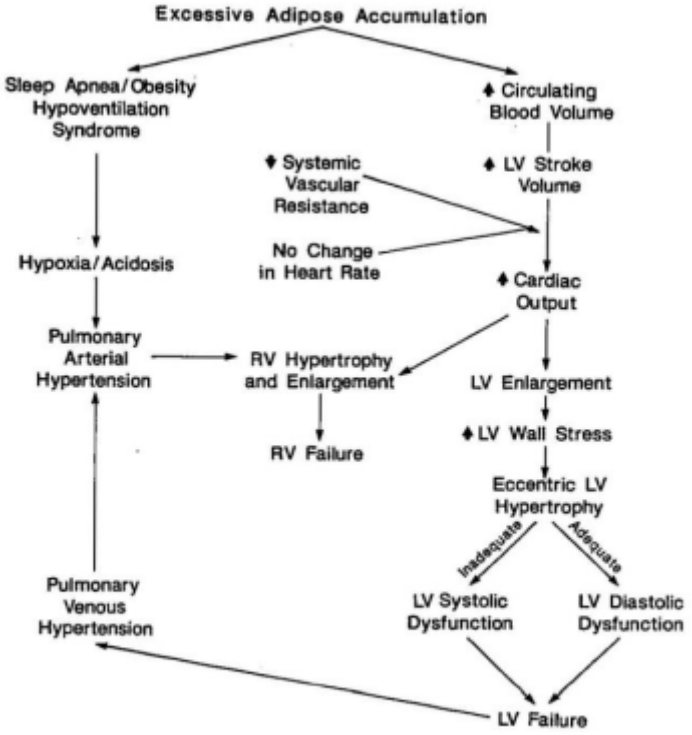


Figure 1. Pathogenesis of congestive heart failure in morbidly obese individuals with and without the sleep apnea/obesity hypoventilation syndrome. LV = left ventricular; RV = right ventricular.

either disorder alone (Table 1). Hypertensive obese individuals often have larger LV dimensions, greater LV wall thicknesses, and higher LV end-diastolic pressures than hypertensive individuals and have LV volumes that are similar to or greater than those of patients with hypertensions. Moreover, LV radius to thickness ratios remain high despite progressive wall thickening, which results in a predisposition to LV systolic dysfunction[50].

The sleep apnea/obesity hypoventilation syndrome, occurring in approximately 5% of morbidly obese individuals, poses a potentially life-threatening complication. (Figure 1)[50].

Treatment strategies for obesity cardiomyopathy involve a multifaceted approach, including weight loss, salt restriction, and the use of diuretics. In certain cases, digitalis and vasodilators may be considered as therapeutic options. It is crucial to address the underlying factors contributing to cardiac dysfunction in obese individuals.

Moreover, central obesity emerges as a probable risk factor for the development of coronary heart disease. Alterations in lipid and insulin metabolism associated with obesity may further contribute to the pathogenesis of coronary heart disease in obese patients. As such, comprehensive management of obesity-related cardiac complications should encompass not only cardiac-specific interventions but also address systemic factors that contribute to cardiovascular risk in the obese population.

REFERENCE

1. Olson LH, Haggstrom J, Henrik DP. Acquired valvular heart disease. In: Ettinger SJ, Feldman EC, editors. Textbook of veterinary internal medicine. 7th edition. St Louis (MO): Saunders Elsevier; 2010. p. 1299–319.
2. Meurs KM. Myocardial disease; canine. In: Ettinger SJ, Feldman EC, editors. Textbook of veterinary internal medicine. 7th edition. St Louis (MO): Saunders Elsevier; 2010. p. 1320–7.
3. Macdonald K. Myocardial disease: feline. In: Ettinger SJ, Feldman EC, editors. Textbook of veterinary internal medicine. 7th edition. St Louis (MO): Saunders Elsevier; 2010. p. 1328–41.
4. Boynosky NA, Stokkin L. Atherosclerosis associated with vasculopathic lesions in a golden retriever with hypercholesterolemia. Can Vet J 2014;55(5):484–8.
5. Jordan E, Kley S, Le NA, et al. Dyslipidemia in obese cats. Domest Anim Endocrinol 2008;35(3):290–9.
6. Hoenig M. The cat as a model for human obesity and diabetes. J Diabetes Sci Technol 2012;6(3):525–33.
7. Verkest KR. Is the metabolic syndrome a useful concept in dogs? A review of the evidence. Vet J 2014;199(1):24–30.
8. Freeman LM, Rush JE, Markwell PJ. Effects of dietary modification in dogs with early chronic valvular disease. J Vet Intern Med 2006;20:1116–26.
9. Thengchaisri N, Wutthiwong T, Kaewmokul S, et al. Abdominal obesity is associated with heart disease in dogs. BMC Vet Res 2014;10(1):131.
10. Freeman LM, Rush JE, Kehayias JJ, et al. Nutrition alterations and the effect of fish oil supplementation. J Vet Intern Med 1998;12:440–8.
11. Atkins CE, Gallo MA, Kurzmann ID, et al. Risk factors, clinical signs, and survival in cats with a clinical diagnosis of hypertrophic cardiomyopathy: 74 cases (1985- 1989). J Am Vet Med Assoc 1992;201(4):613–8.
12. Yang VK, Freeman LM, Rush JE. Comparisons of morphometric measurements and serum insulin-like growth factor concentration in healthy cats and cats with hypertrophic cardiomyopathy. Am J Vet Res 2008;69(8):1061–6.
13. Van Vliet BN, Hall JE, Mizelle HL, et al. Reduced parasympathetic control of heart rate in obese dogs. Am J Physiol 1995;269(2 Pt 2):H629–37.
14. Truett AA, Borne AT, Poincot MA, et al. Autonomic control of blood pressure and heart rate in obese hypertensive dogs. Am J Physiol 1996;270(3):R541–9.

15. Boutheguard JC, Kelly M, Clety N, et al. Effects of weight loss on heart rate normalization and increase in spontaneous activity in moderately exercised overweight dogs. Int J Appl Res Vet Med 2009;7(4):153–64.

16. Mehlman E, Bright JM, Jeckel K, et al. Echocardiographic evidence of left ventricular hypertrophy in obese dogs. J Vet Intern Med 2013;27(1):62–8.

17. Radin MJ, Sharkey LC, Hylucross BJ. Adipokines: a review of biological and analytical principles and an update in dogs, cats and horses. Vet Clin Pathol 2009;38/2:126–56.

18. Engeli S, Schling P, Gorzelniak K, et al. The adipose-tissue renin-angiotensinaldosterone system: role in the metabolic syndrome? Int J Biochem Cell Biol 2003;35:807–25.

19. Barton M, Carmona R, Ortmann J, et al. Obesity-associated activation of angiotensin and endothelin in the cardiovascular system. Int J Biochem Cell Biol 2003; 35:826–37.

20. Henegar JR, Bigler SA, Henegar LK, et al. Functional and structural changes in the kidney in the early stages of obesity. J Am Soc Nephrol 2001;12:1211–7.

21. Adolphe JL, Silver TI, Childs H, et al. Short-term obesity results in detrimental metabolic and cardiovascular changes that may not be reversed with weight loss in an obese dog model. Br J Nutr 2014;112:647–56.

22. Rocchini AP, Moorehead C, Wentz E, et al. Obesity-induced hypertension in the dog. Hypertension 1987;9(6):11164–8.

23. De Paula RB, da Silva AA, Hall JE. Aldosterone antagonism attenuates obesityinduced hypertension and glomerular hyperfiltration. Hypertension 2004;43:41–7.

24. Pinhall CS, Lopes A, Torres DB, et al. Time-course morphological and functional disorders of the kidney induced by long-term high-fat intake in female rats. Nephrol Dial Transplant 2013;28:2464–76.

25. Engli S, Bohnke J, Gorzelniak K, et al. Weight loss and the renin-angiotensinaldosterone system. Hypertension 2005;45:356–63.

26. Pena C, Suarez L, Bautista-Castano I, et al. Effects of low-fat high-fibre diet and mitratapide on body weight reduction, blood pressure and metabolic parameters in obese dogs. J Vet Med Sci 2014;176(9):1305–8.

27. Frigolet ME, Torres N, Tovar AR. The renin-angiotensin system in adipose tissue and its metabolic consequences during obesity. J Nutr Biochem 2013;24(12): 2003–15.

28. Luo H, Wang X, Chen C. Oxidative stress causes imbalance of renal renin angiotensin system (RAS) components and hypertension in obese Zucker rats. J Am Heart Assoc 2015;4(2):102–10.

29. Grant R, Vester- Boler BM, Ridge TK, et al. Adipose tissue transcriptome changes during obesity development in female dogs. Physiol Genomics 2011;43(6): 295–307.

30. Hoenig M, Pach N, Thomaseth K, et al. Cats differ from other species in their cytokine and antioxidant enzyme response when developing obesity. Obesity (Silver Spring) 2013;21(9):E407–14.

31. Ishioka K, Omachi A, Sagawa M, et al. Canine adiponectin:cDNA structure, mRNA expression in adipose tissues and reduced plasma levels in obesity. Res Vet Sci 2006;80(2):127–32.

32. Hyung-Jin P, Sang-Eun L, Jung-Hyun O. Leptin, adiponectin and serotonin levels in lean and obese dogs. BMC Vet Res 2014;10(1):113–9.

33. Park HJ, Lee SE, Kim HB, et al. Association of obesity with serum leptin, adiponectin, and serotonin and gut microflora in beagle dogs. J Vet Intern Med 2015; 29(1):43–50.

34. Verkest KR, Rose FJ, Fleeman LM, et al. Adiposity and adiponectin in dogs: investigation of causes of discrepant results between two studies. Domest Anim Endocrinol 2011;41:35–41.

35. Hoenig M, Thomaseth K, Waldron M, et al. Insulin sensitivity, fat distribution, and adipocytokine response to different diets in lean and obese cats before and after weight loss. Am J Physiol Regul Integr Comp Physiol 2007;292(1):R227–34.

36. Ryan VH, German AJ, Wood IS, et al. Adipokine expression and secretion by canine adipocytes: stimulation of inflammatory adipokine production by LPS and TNF alpha. Pflugers Arch 2010;460(3):603–16.

37. Shibata R, Izumiya Y, Sato K, et al. Adiponectin protects against the development of systolic dysfunction following myocardial infarction. J Mol Cell Cardiol 2007;75: 1065–74.

38. Fesus G, Dubrovska G, Gorzelnaik K, et al. Adiponectin is a novel humoral vasodilator. Cardiovasc Res 2007;75:719–27.

39. Ouchi N, Ohishi M, Kihara S, et al. Association of hypoadiponectinemia with impaired vasoreactivity. Hypertension 2003;42:231–4.

40. Rocchini AP, Yang JQ, Gokee A. Hypertension and insulin resistance are not directly related in obese dogs. Hypertension 2004;43(5):1011–6.

41. Yokota T, Oritani K, Takahasi I, et al. Adiponectin, a new member of the family of soluble defence collagens, negatively regulates the growth of myelomonocytic progenitors and the functions of macrophages. Blood 2000;96:1723–32.

42. Pelosi A, Rosenstein D, Abood SK, et al. Cardiac effect of short-term experimental weight gain and loss in dogs. Vet Rec 2013;172(6):153–60.

43. Vitger AD, Stallknecht BM, Nielsen DH, et al. Integration of a physical training program in a weight loss plan for overweight pet dogs. J Am Vet Med Assoc 2016; 248(2):174–82.

44. Manens J, Ricci R, Damoiseaux C, et al. Effect of body weight loss on cardiopulmonary function assessed by 6-minute walk test and arterial blood gas analysis in obese dogs. J Vet Intern Med 2014;28:371–8.

45. Horwich TB, Fonarow GC, Hamilton MA, et al. The relationship between obesity and mortality in patients with heart failure. J Am Coll Cardiol 2001;38(3):789–95.

46. Davos CH, Doehner W, Rauchhaus M, et al. Body mass and survival in patients with chronic heart failure without cachexia: the importance of obesity. J Card Fail 2003;9(1):29–35.

47. Slupe JL, Freeman LM, Rush JE. Association of body weight and body condition with survival in dogs with heart failure. J Vet Intern Med 2008;22:561–5.

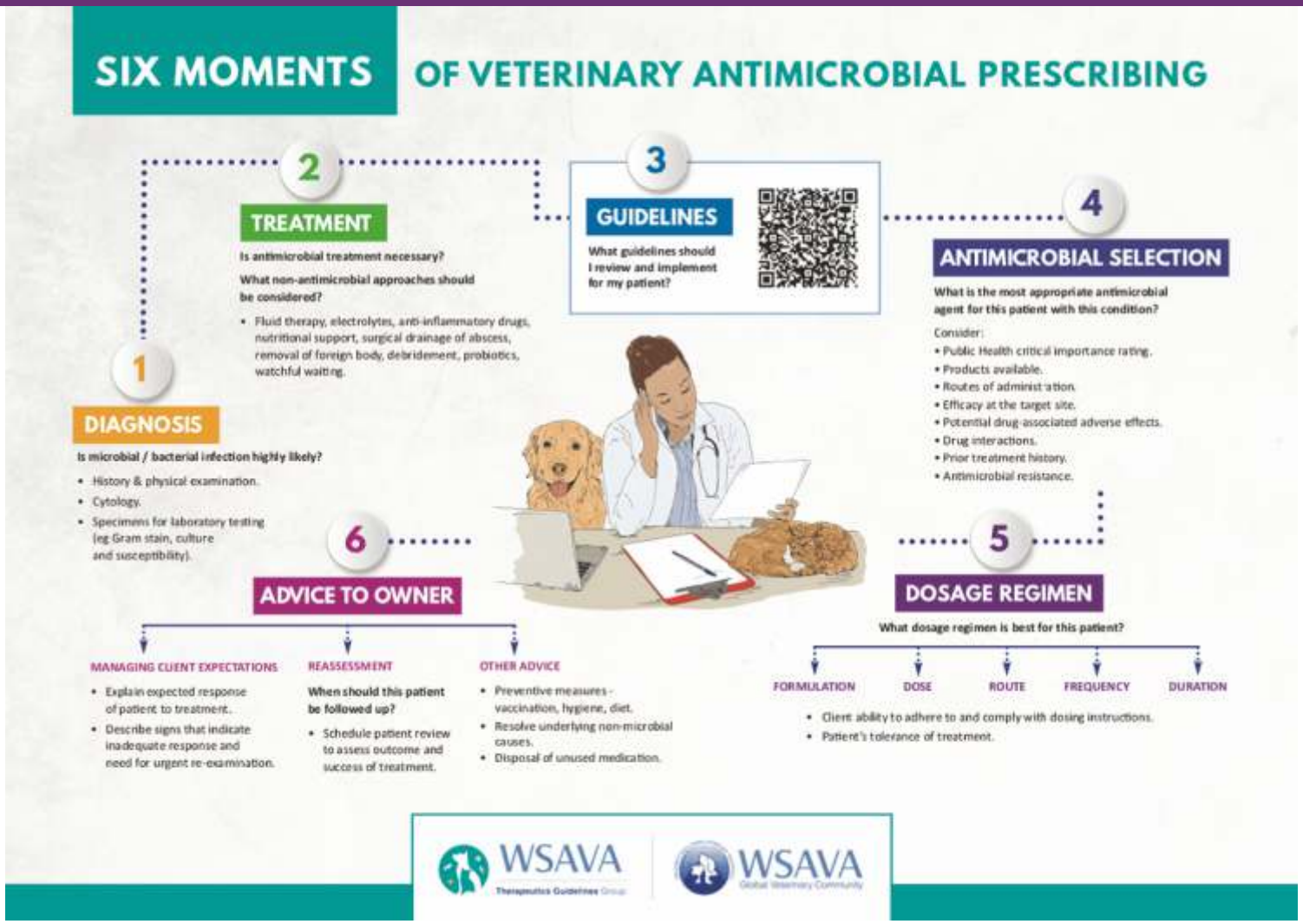
48. Finn E, Freeman LM, Rush LE, et al. The relationship between body weight, body condition and survival in cats with heart failure. J Vet Intern Med 2010;24: 1369–74.

49. Curtis JP, Selter JG, Wang Y, et al. The obesity paradox. Arch Intern Med 2005; 165:55–61.

50. Alpert, M. A., & Hashimi, M. W. (1993). Obesity and the heart. The American journal of the medical sciences, 306(2), 117-123.

51. Chandler, M.L., 2016. Impact of obesity on cardiopulmonary disease. Veterinary Clinics: Small Animal Practice, 46(5), pp.817-830.

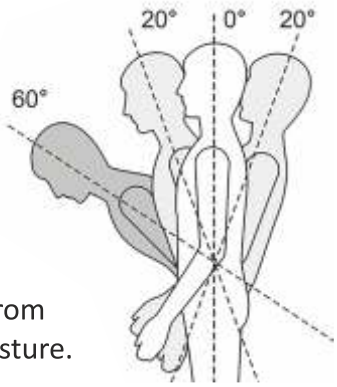
WSAVA Guide lines for Use of Antimicrobial



Occupational Hazards in Veterinary Profession

(Short note on Awkward Posture)

Veterinary professionals in India have to struggle daily in their professional lives which takes a toll on their personal, physical, mental, economic and social wellbeing. With more and more veterinarians joining the work force in private sector the competition to retain clients and work extra hours to keep veterinary facilities open 24x 7 adds to the work pressure. Other documented hazards include



1. Working with animals, especially uncooperative animals pose risks from animal bites and other physical injuries. So proper design of facilities/ manpower is needed to handle such patients.
2. Musculoskeletal disorders are common among veterinarians. Disorders result from sudden or sustained exposure to force, vibration, repetitive motion, or awkward posture.

One of the important Hazzard is awkward postures that can cause muscle fatigue and injury.

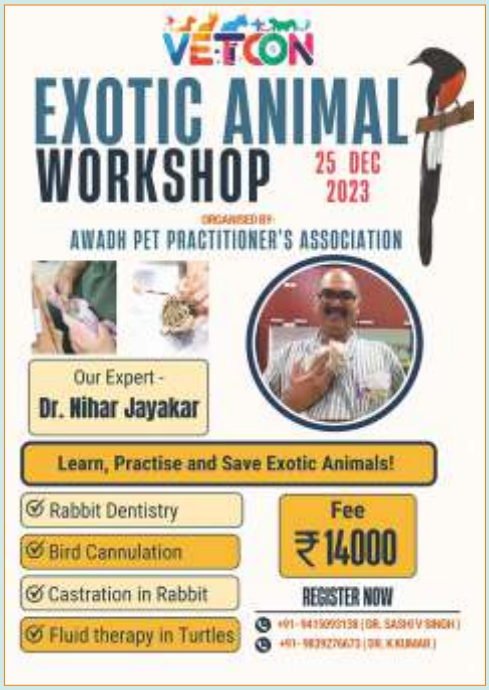
a) Working with the hand(s) above the head, or the elbow(s) above the shoulder, for extended time periods.
b) Working with the neck, back or wrist(s) bent more than 30 degrees for extended time periods.
c) Squatting or kneeling for extended time periods.
d) Sustained position for extended time periods.

Proud Moment for PPAM Members

First Vet CE on live heart simulator by PPAM and VIVALDIS at IMMAST

Dr. Sangeeta V. Shah

Dr. Nihar Jayakar



Dr. Nihar Jayakar

Dr. Jairam spoke on 14.10.2023 at Bombay Veterinary College at ICSR, MAFSU organised AGRI fair.

Dr. Shriniwas. V. Vishwasrao conducted CE in Lucknow on 14.10.23 and in Bangalore on 17.12.2023 in Association with PPAK (Pet Practitioner Association of Karnataka, Meril Veterinary and Vetina.

CE at Bangalore, PPAK

CE at Bangalore, PPAK

Dr. S. V. Vishwasrao was bestowed the A. P. Singh Life time achievement award for his work on Veterinary Radiology and Imaging in the ISVS (Indian Society for Veterinary Surgery) congress held in Bhubaneshwar, Odisha held from 6th to 8th December 2023.

LUCKNOW

BHUBANESHWAR

Nipah virus.
What veterinarians must know.

Nipah virus disease is an infectious zoonotic diseases caused by Nipah viruses (NiV). This disease causes respiratory and occasionally nervous signs in susceptible animals such as pigs and horses and could have devastating zoonotic potential.

Between 12 and 15 September 2023, a total of six laboratory-confirmed cases of Nipah virus infection in humans were reported by the State Government of Kerala. All confirmed cases were reported within the Kozhikode district of Kerala.

Nipah virus infection is an emerging bat-borne zoonotic disease transmitted to humans through infected animals (such as bats and pigs) or food contaminated with saliva, urine, and excreta of infected animals. It can also be transmitted directly from person to person through close contact with an infected person.

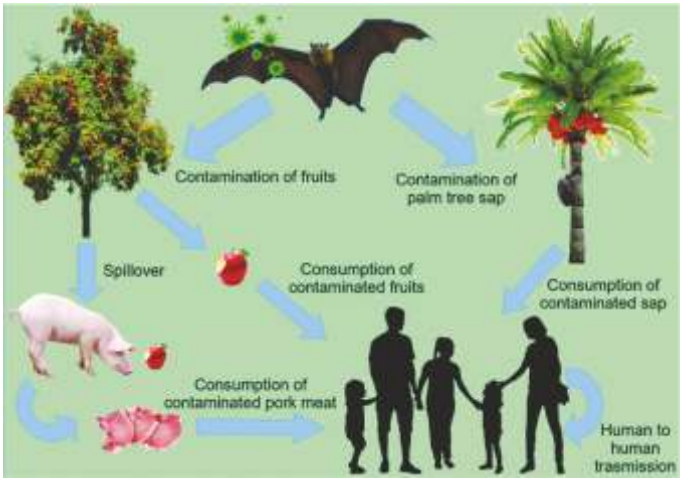
Reducing the risk of bat-to-human transmission

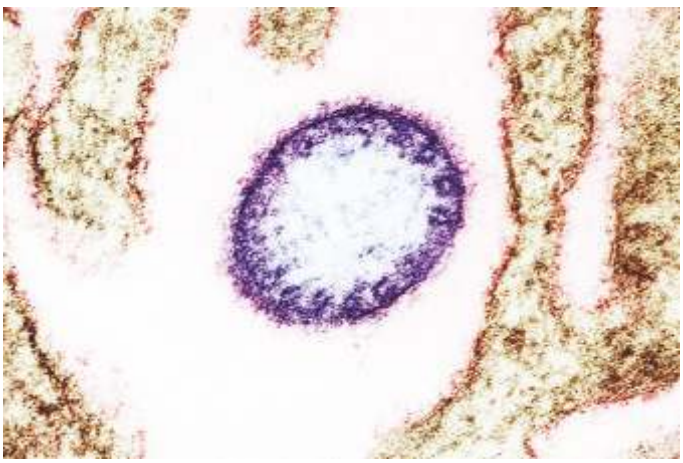
Freshly collected date palm juice should be boiled, and fruits should be thoroughly washed and peeled before consumption. Fruits with signs of bat bites should be discarded. Areas where bats are known to roost should be avoided. The risk of international transmission via fruit or fruit products (such as raw date palm juice) contaminated with urine or saliva from infected fruit bats can be prevented by washing them thoroughly and peeling them before consumption.

Reducing the risk of animal-to-human transmission

Natural infection in domestic animals has been described in farming pigs, horses, and domestic and feral cats. Gloves and other protective clothing should be worn while handling sick animals or their tissues and during slaughtering and culling procedures. As much as possible, people should avoid being in contact with infected pigs. In endemic areas, when establishing new pig farms, consideration should be given to the presence of fruit bats in the area and in general, pig feed and pig sheds should be protected against bats when feasible.

Samples taken from animals with suspected Nipah virus infection should be handled by trained staff working in suitably equipped laboratories.





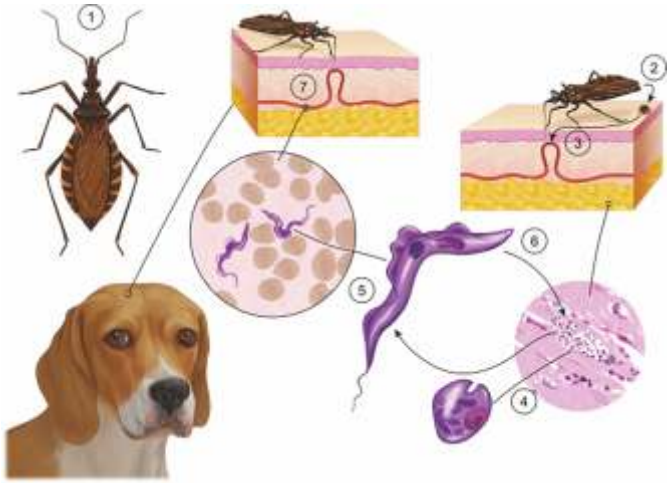
Canine Trypanosomiasis

Canine trypanosomiasis, caused by protozoans of the genus Trypanosoma, is divided into two primary types: the American form (Chagas disease), due to Trypanosoma cruzi infection, and the African form (sleeping sickness or surra), provoked by Trypanosoma evansi. The trypanosome species that infect dogs include T. evansi, which presents the salivary form (transmitted by saliva from the vector), and T. cruzi, which presents the stercorary form (transmitted by feces from the vector).

The acute phase can be observed in young dogs between five and six months old. The affected animals develop generalized infection with extensive lesions, mainly in the myocardium and central nervous system. The acute phase is further characterized by anorexia, generalized lymphadenopathy, diarrhea, myocarditis, and sudden death, which can occur due to serious cardiac arrhythmia.

It is more difficult to diagnose the acute phase of the disease in adult dogs. The indeterminate chronic form occurs eight to 36 months after initial infection and is characterized by ventricular arrhythmias and myocardial dilation. Signs of cardiac insufficiency are initially seen on the right side, with progression to biventricular insufficiency. Important clinical signs in naturally infected dogs include: intermittent fever, progressive anemia, anorexia, cachexia, edema of the head, pharynx, and limb, lymphadenopathy, rear limb paralysis, ataxia, a lack of coordination, tonic-clonic seizures, opisthotomus, and corneal opacity.

Diagnosis techniques is based on DNA detection by PCR, it has proved more sensitive than conventional parasitological methods. However, its high cost and complexity are still drawbacks to its routine use in diagnosis



Treatment consists of Diminazene aceturate and has been shown to be an effective treatment for T. evansi at a dose of 7 mg/kg on the first day and 3.5 mg/kg on the following day.

Using Pesticides on Pets. As Veterinarians we owe a responsibility to Guide Pet Parents.

Pet parents use pesticides on their animals to control external parasites. They can be very beneficial, but they can cause harm to pets, especially if they are not used according to the label directions.

As veterinarians we need to instruct our pet parents appropriately:

1. Follow the label carefully. More is not better! Over-treating animals can lead to illness or death.
2. The prescribed dose for most topical formulations is determined by the animal's weight.
3. Dog products should never be used on cats unless labelled for cats.
4. Products designed for adult pets should never be used on kittens or puppies unless the label states that the product may be used on younger animals.
5. Pet parents wear protective clothing as required by the label to minimize exposure.
6. It's a common mistake to use flea and/or tick shampoo before using another insecticide on the animal's fur. Unless the label directions for the two products indicate they can be used together, select only one of the products for use.

Lice

Flea

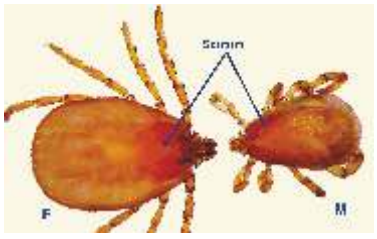
Tick

Mite

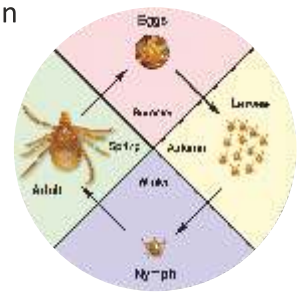
7. Many products require treated animals to be separated from other pets after treatment to prevent touching and licking.
8. Beware of counterfeit products, which may not be properly packaged, labelled or formulated correctly.

Dog Ticks. Let us refresh our knowledge.

Most ticks go through four life stages: egg, six-legged larva, eight-legged nymph, and adult. After hatching from the eggs, ticks must eat blood at every stage to survive.



Two types of ticks Hard ticks and Soft ticks. Ticks have three different life cycles. Depending on the species, can either possess a one-host life cycle, two-host life cycle, or three-host life cycle. One of the most common dog ticks seen in India is the brown dog tick. Studies indicate that Rhipicephalus ticks are the most common arthropod vectors. The most prevalent canine tick borne disease is Hepatozoon canis, followed by E. canis, M. haemocanis and A. platys. Numerous studies indicate that PCR is more sensitive in detecting blood pathogens compared with microscopic blood film examination.



DROOLS ARTICLE

A Comprehensive Review of Hepatic Wet Food in managing Liver disorders in Dogs

Dr. Pooja Chitteni
Veterinary Product Executive (Specialist)

1. Introduction:

Canine hepatic issues have become a prevalent concern among dog owners, necessitating a closer look at dietary interventions to support liver health. The liver plays a central role in nutrient metabolism and because of this, the requirements for specific nutrients may change following onset of liver failure. It also plays vital role in synthesizing essential components such as plasma proteins, clotting factors, and bile salts. The liver is vital for digestion and nutrient storage. When liver health is compromised, issues like poor appetite and nutrient absorption arise. Nutritional therapy is key for managing liver conditions. Although there are differences amongst all nutritional approaches for managing hospitalized patients with different hepatopathies in dogs , they can be broadly classified according to protein intolerance. While end-stage cirrhosis and congenital and acquired vascular shunting may necessitate significant protein restriction, some hepatopathies, such as cholangitis, triaditis, and hepatic lipidosis, do not usually require it. This review focuses on the potential benefits of hepatic wet food for dogs, exploring its role in mitigating symptoms and promoting overall liver well-being.

Key words: Liver, Nutrient, Hepatopathies, Wet food, Dog.

2. Hepatic Ailments in Dogs:

Common hepatic issues in dogs include hepatitis (liver inflammation), cirrhosis (chronic scarring and loss of function), hepatic lipidosis (accumulation of excess fat in the liver), cholangitis (bile duct inflammation), triaditis (simultaneous inflammation of the liver, pancreas, and intestines), vascular shunting (abnormal blood flow bypassing the liver), congenital liver disorders (birth defects affecting the liver), liver tumors (benign or malignant growths), and toxin-induced liver damage. Tailoring nutrition to the specific hepatic issue is crucial for supporting the liver's function and aiding in the overall well-being of the dog.

3. Symptoms and Outcomes:

Symptoms and outcomes commonly observed in hepatic ailments in dogs include lethargy, jaundice (yellowing of the eyes and gums), weight loss, changes in appetite (either increased or decreased), vomiting, diarrhea, increased thirst, and abdominal discomfort. In more severe cases, hepatic diseases can progress to liver failure, leading to advanced symptoms such as neurological abnormalities, fluid accumulation in the abdomen (ascites), and an overall decline in the dog's health. Early detection and prompt intervention are crucial to mitigate these symptoms, prevent complications, and improve the prognosis for dogs with hepatic issues.

4. Nutritional Considerations for Hepatic health:

Affected animals should be fed small, frequent meals throughout the day, which may also help increase energy intake in finicky or hyporectic patients. The best diet for any patient with Hepatic disorders will vary from animal to animal. A complete diet history can facilitate selecting a diet, especially if protein restriction is indicated. Once a diet has been selected, the animal should be monitored frequently for adverse responses to the food; food intake; body weight and condition; and laboratory parameters.

4.1 Protein regulation:

Adapting protein intake to the individual hepatic condition. While some cases may necessitate moderation to ease the liver's workload, others may tolerate a normal protein diet. Hepatic disease is characterized by altered liver regulation of amino acid homeostasis and possibly reduced glycogen stores. Appropriate dietary protein can help close the energy deficit

caused by low glycogen stores in liver disease and facilitate a quicker switch from amino acids to glyconeogenesis (Silk *et al.*, 1991). Therefore, it is crucial to provide a diet that has a slightly higher protein content made up of high-quality protein when managing chronic hepatopathies. In animals that respond well to protein restriction, consideration should be given to slowly increasing the amount of protein in the diet until clinical signs recur or the animal is consuming protein levels equivalent to an adult maintenance food (about 20–30% protein calories in dogs and about 30–40% protein calories in cats).

4.2 Fat moderation:

However, the fat content of foods should be limited in patients with potential cholelithiasis or cholestasis. High dietary cholesterol, implicated in gallstone formation, requires limiting fat intake in hepatobiliary disease. Reduced lipid metabolism may lead to diarrhea, emphasizing the importance of a low-fat diet (10% DM fat for dogs, <15% for cats). Shetland sheepdogs with gallbladder issues improved on such diets (LaFlamme, 2000).

4.3 Carbohydrate and fibre:

Hepatogenic hypoglycemia is more common in dogs with cirrhosis, congenital PSS (Portosystemic shunt), fulminant hepatic failure, and extensive liver neoplasia when it comes to veterinary patients (Marks *et al.*, 1994). Frequent small meal feedings could potentially help avoid hypoglycemia. By enhancing colonic transit and preventing constipation, the addition of soluble and insoluble fibers reduces the amount of time that toxins are absorbed from the colon (Meyer *et al.*, 2010).

4.4 Copper management:

Abnormal hepatic copper concentrations are often observed as a pathologic feature in hepatic disease. Breeds associated with primary copper-associated hepatopathies include Skye terriers, West Highland white terriers, Doberman pinschers, Labrador retrievers and Bedlington terriers. Managing elevated hepatic copper levels reduces mitochondrial damage and lipid peroxidation in hepatocytes (Sokol *et al.*, 1989). Traditional treatments involve lowering total dietary copper, increasing dietary zinc, and implementing chelation therapy (Hoffmann *et al.*, 2009).

4.5 L-carnitine role:

The use of L-carnitine is often implemented as it has been shown that it may improve whole body fat metabolism and help prevent hepatic lipidosis and ketosis even though liver and serum carnitine concentrations in hepatic lipidosis are not depleted (Blanchard *et al.*, 2002).

4.6 Cobalamin (vitamin B12)

A degenerative liver disease has been diagnosed in young Beagles as a result of hereditary cobalamin malabsorption from a mutation in the cubilin gene (Kook *et al.*, 2014). In dogs with liver disorders, 37.5% had hypercobalaminaemia and 26.7% had elevated serum methylmalonic acid (MMA) concentrations (Fry *et al.*,2013)

4.7 Low Sodium Diets:

Addressing fluid retention, a common consequence in hepatic diseases, by implementing diets with restricted sodium content.

4.8 Omega 3 fatty acids:

A diet containing omega-3 fatty acids is crucial in managing liver disorders in dogs due to their anti-inflammatory and antioxidant properties. Omega-3s support hepatic function, reduce oxidative stress, and potentially improve lipid metabolism.

4.9 Supplemental Support:

Administering vitamin and mineral supplements as needed to compensate for potential deficiencies associated with hepatic dysfunction.

4.10 Continuous Veterinary Monitoring:

Regular veterinary check-ups are indispensable for assessing the dog's nutritional status, evaluating the progression of hepatic diseases, and making necessary adjustments to the

dietary plan. This collaborative approach ensures a tailored and effective nutritional strategy for dogs with hepatic conditions.

4.11 Role of hepatic wet food:

Feeding wet food to dogs with hepatic diseases is a strategic dietary choice aimed at supporting their liver health. Its moisture-rich content helps address dehydration, a common concern in hepatic patients. The formulation of these wet foods often includes high-quality proteins, easily digestible carbohydrates, and essential nutrients tailored to support liver function. Additionally, the palatable nature of wet food can entice dogs with reduced appetite, encouraging them to consume necessary nutrients Drools VET-PRO Hepatic gravy is a specialized prescription diet for canine hepatic issues, prioritizing liver health. With high-quality protein, controlled carbs, omega-3 fatty acids, and vitamin B12, it supports liver and muscle well-being. The formula limits copper, preventing excess buildup, and lowers sulfur, reducing ammonia production. Meticulously crafted, it eases the liver's load, serving as a cornerstone in holistic hepatic care for dogs.

5. Conclusion:

In conclusion, a nuanced approach to nutrition is crucial in managing canine hepatic issues. Tailoring diets to specific conditions, monitoring protein and fat intake, managing copper levels, and incorporating omega-3 fatty acids are vital considerations. The strategic use of hepatic wet food, such as VET-PRO Hepatic gravy, not only supports liver health but also ensures palatability, making it an ideal choice for dogs with reduced appetite. The collaborative effort of continuous veterinary monitoring and specialized nutrition, such as the VET-PRO Hepatic gravy diet, proves instrumental in enhancing the overall prognosis and well-being of dogs facing hepatic conditions.

6. References:

Blanchard, G., Paragon, B. M., Milliat, F., & Lutton, C. (2002). Dietary L-carnitine supplementation in obese cats alters carnitine metabolism and decreases ketosis during fasting and induced hepatic lipidosis. *The Journal of nutrition, 132*(2), 204-210.

Fry, J. K., Burney, D. P., Lidbury, J. A., Steiner, J. M., Suchodolski, J. S., Hottinger, H. A., ... & Mitchell, M. A. (2013, May). Evaluation of cobalamin status in dogs with hepatic disease. In *Journal Of Veterinary Internal Medicine* (Vol. 27, No. 3, Pp. 716-717). 111 River St, Hoboken 07030-5774, Nj Usa: Wiley-Blackwell.

Hoffmann, G., Jones, P. G., Biourge, V., Van Den Ingh, T. S. G. A. M., Mesu, S. J., Bode, P., & Rothuizen, J. (2009). Dietary management of hepatic copper accumulation in Labrador Retrievers. *Journal of veterinary internal medicine, 23*(5), 957-963.

Kook, P. H., Drögemüller, M., Leeb, T., Howard, J., & Ruetten, M. (2014). Degenerative liver disease in young Beagles with hereditary cobalamin malabsorption because of a mutation in the cubilin gene. *Journal of veterinary internal medicine, 28*(2), 666.

Laflamme, D. P. (2000). Nutritional management of liver disease. *Kirks Current Veterinary Therapy, 13*, 693-696.

Marks, S. L., Rogers, Q. R., & Strombeck, D. R. (1994). Nutritional Support In Hepatic-Disease. 1. Metabolic Alterations And Nutritional Considerations In Dogs And Cats. *Compendium On Continuing Education For The Practicing Veterinarian, 16*(8), 971.

Meyer HP, Twedt DC, Roudebush P, Dill-Mackay E. (2010) Hepatobility disease. In Small Animal Clinical Nutrition, 5th ed. Hand M.S, Thatcher C.D., Remillard R.L., Roudnebush P, and Novotny B., eds. Mark Morris Institute. Topeka, Kansas, USA. pp:1155-1194

Silk, D. B., O'Keefe, S. J., & Wicks, C. (1991). Nutritional support in liver disease. *Gut, 32*(Suppl), S29.

Sokol, R. J., Devereaux, M. W., Traber, M. G., & Shikes, R. H. (1989). Copper toxicity and lipid peroxidation in isolated rat hepatocytes: effect of vitamin E. *Pediatric research*, 25(1), 55-62.

Dr. Snehkant G. Swali Founding member of PPAM left for his heavenly abode 01/01/2024. He was the first privately practicing veterinarian in Mumbai. When Dr. Swali initiated PPAM as an organization it was with only 32 members.

Dr. Snehkant G. Swali graduated in 1964 and in his own words would describe his journey in veterinary profession from horse doctor to pet practitioner. Dr. Swali was giving Hon service to Panjrapole for more than 50 years.

In words of Dr. Swali this veterinary profession will give swarth and parmarth both. He will be greatly missed by the PPAM family, hundreds of pets, pet parents and veterinarians. Dr. Swali sir we at PPAM will always carry your memory at Heart.

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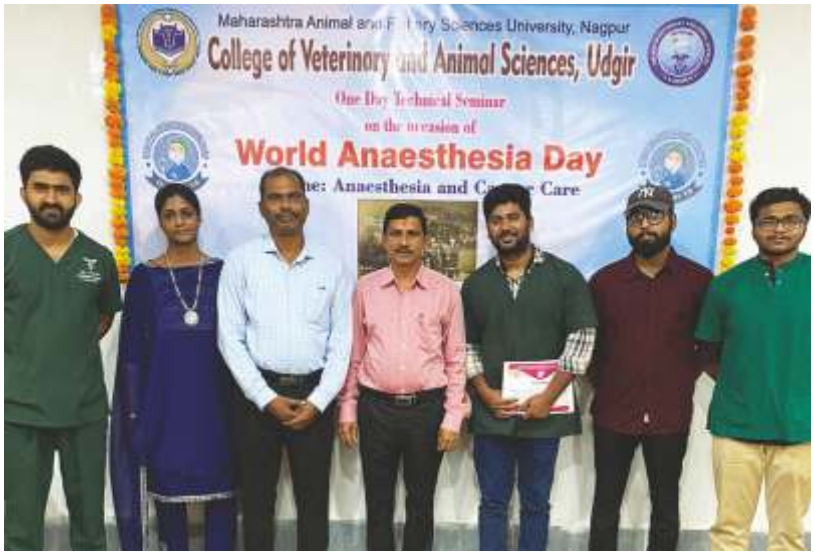

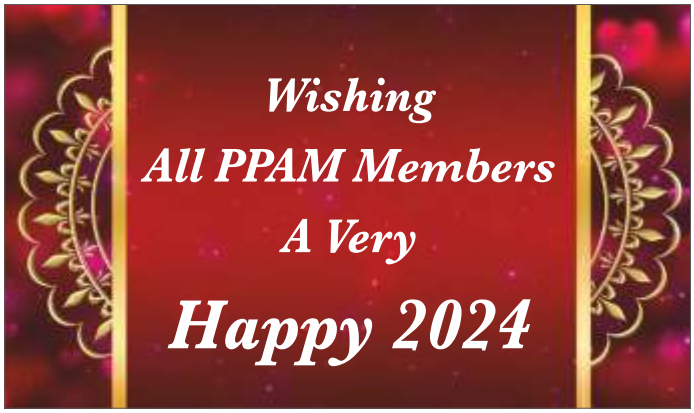

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